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Acute Simultaneous Thrombotic Occlusion of Multiple Coronary Arteries in Acute Myocardial Infarction: A Case Report

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Abstract

Introduction: Simultaneous multiple coronary artery thrombosis is a rare finding in ST segment elevation myocardial infarction (STEMI), and has a high mortality rate.

Case Presentation: We report a case of myocardial infarction with cardiogenic shock, left bundle branch block, and multiple ST segment elevation on the electrocardiogram and thrombotic occlusion of the left circumflex, optus marginal, and left anterior descending arteries on emergency coronary angiography. Thrombus aspiration was performed at left circumflex, optus marginal, and left anterior descending arteries.

Conclusions: In patients with STEMI, multiple coronary thrombosis is unusual and associated with high patient mortality.

Keywords: Simultaneous Coronary Thrombosis, Acute Myocardial Infarction, Multiple Thrombosis, Acute ST Elevation Myocardial Infarction and Cardiogenic Shock

1. Introduction

Simultaneous obstruction of the coronary arteries in patients with acute myocardial infarction is uncommon. These patients are usually very ill, and many of them die before reaching the hospital. In most patients with acute myocardial infarction, one of the atherosclerotic coronary arteries, with rupture of atheromatous plaques and clot formation, is blocked. Multiple coronary artery occlusions is a rare phenomenon. Therefore, its early diagnosis and rapid and proper treatment are effective in reducing mortality.

The mechanism of simultaneous multiple coronary occlusion is not clear. Several factors such as essential thrombocythemia, multivessel spasm, hyper coagulability state and abuse of cocaine should be considered. Moreover, traditional risk factors for coronary heart disease such as diabetes, hyperlipidemia, and smoking also help obstruction. Capability of multiple coronary emboli due to the endocarditis, clot emboli from cardiac chamber in patients with no risk factors for coronary artery disease, and paradoxical emboli through an intracardiac shunt must be considered. Thus, patients with multiple culprit arteries in the background of ST elevation myocardial infarctions represent an unusual population with high rates of cardiogenic shock, with no definite cause (1).

2. Case Presentation

In this case report, a 56-year-old female involved in a recent car accident and with a history of hypertension was referred to the emergency department with retrosternal chest pain and dyspnea while sleep. The first vital signs showed irregular rapid heart rhythm and rate and wide QRS compatible with left bundle branch block and atrial fibrillation, a blood pressure of 110/80 mmhg, and a respiratory rate of 20/minute. The results of the electrocardiogram revealed atrial fibrillation and left bundle branch block and ST elevation at I, avl, II, III, avf, and V1 to V6. Atrial fibrillation was converted to sinus rhythm spontaneously (Figure 1); echocardiogram showed 30%, EF. In the catheterization laboratory, the patient was unresponsive on arrival, she had respiratory distress, tachypnea, and blood pressure of 75 mmhg. After prep and drep from right femoral artery, coronary angiography was performed and showed large thrombus with flow limiting at the proximal part of the left circumflex artery, mid part of the large bifurcated optus marginal artery, and mid part of left anterior descending artery (Figure 2A - 2C). The patient was scheduled for coronary thrombectomy, 5000 IU heparin, and glycoprotein IIb/IIIa inhibitor integrilin was injected. After left coronary artery engagement with 6 French left judkins catheter and wiring with 0.014 inches BMW, multiple thrombectomy was performed with export catheter, and then, coronary angiography was performed, revealing a successful resolution of thrombus and maintenance of intravenous heparin; integrilin was continued and she was placed on dual anticipate therapy with aspirin and clopidogrel. The patient was transferred into the critical care unit. Lab data showed the following information:

hemoglobin: 13.6 mg/dL; white cell count: 11,400 per microliter; platelet count: 75,000 per microliter; creatinine: 1.3 mg/dL; troponin: 38.68 ng/dL; creatinine kinase: 4169 IU/L: and CK MB 520/L; glucose: 338 mg/dL; cholesterol: 162 mg/dL; low density lipoprotein cholesterol: 104 mg/dL; high density lipoprotein cholesterol: 35 mg/dL; and triglyceride: 193 mg/dL.

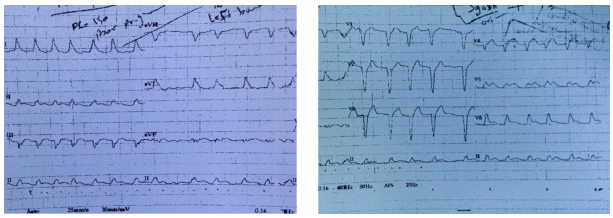

Figure 1. Electrocardiogram showed atrial fibrillation with rapid ventricular response and new left bundle branch block

Figure 2. Coronary angiography showed multiple simultaneouse thrombosis in left circumflex artery (A) and left anterior descending artery (B) and abtus marginal artery (C)

3. Discussion

Most patients with STEMI have 1 culprit lesion in coronary artery that leads to acute occlusion of that coronary artery. Occasionally, more than 1occluded artery may be present; however, usually only one is an acute event, the others are chronic occlusions (2). Simultaneous thrombosis of multiple epicardial coronary arteries is an uncommon clinical finding in STEMI (3), and is usually associated with cardiogenic shock and dangerous arrhythmia (4). Facilitating factors to create multiple coronary thrombosis includes coronary vasospasm (5, 6), cocaine abuse (7), higher likelihood of thrombosis such as in heparin induced thrombocytopenia (8), antithrombin III deficiency (9), idiopathic thrombocytopenic purpura (10), hormonal therapy such as tamoxifen (11) as well as thrombophilias such as antiphospholipid antibodies, factor V Leiden deficiency, essential thrombocythemia, and hyper- homocysteinemia (12-16). Coronary embolism from cardiac chamber is another source of the simultaneous multiple coronary artery thrombosis. Multiple coronary thrombosis was demonstrated in 10% of the patients who died from acute myocardial infarction (17). Multiple simultaneous coronary occlusions in acute myocardial infarction always lead to very serious illness (1). In previous reports, more than 50% of patients presented with cardiogenic shock with poor prognosis, and had a high mortality rate. However, in clinical practice, it is particularly uncommon for multiple coronary thrombosis to be diagnosed because these patients tend to present with cardiogenic shock or a rapid and fatal course (4). Our patient presented with STEMI and cardiogenic shock, and her coronary angiography showed multiple coronary thrombosis. She had atrial fibrillation at initial ECG that represented 1 source for atrial clot formation and coronary embolism. Also, the patient had a history of car accident and head trauma that predisposed her for catecholamine release and formation of multiple thrombosis in coronary arteries. According to thrombocytopenia, heparin induced thrombocytopenia, and idiopathic thrombocytopenia purpura should also be considered. After coronary angiography, percutaneous coronary intervention with thrombectomy was performed and following aspiration of a large thrombus, coronary blood flow was restored. Angiography results revealed coronary arteries without any evidence of plaque or stenosis requiring coronary stenting.

One reason for patients with STEMI and cardiogenic shock is simultaneous multiple coronary artery thrombosis with a high mortality rate.

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